09/775,856

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=> d his

     (FILE 'HOME' ENTERED AT 02:35:11 ON 20 OCT 2003)

     FILE 'HCAPLUS' ENTERED AT 02:35:20 ON 20 OCT 2003
L1            39 S (IPG OR INOSITOL(2A)PHOSPHOGLYCAN?) (P) (LIVER? OR PLACENTA?)
L2             7 S L1 AND ANTIBOD?

     FILE 'STNGUIDE' ENTERED AT 02:38:43 ON 20 OCT 2003

=>
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=> s (IPG or inositol(2a)phosphoglycan?) (p) (liver? or placenta?)
           423 IPG
         34587 INOSITOL
           152 PHOSPHOGLYCAN?
        497577 LIVER?
         45112 PLACENTA?
L1          39 (IPG OR INOSITOL(2A)PHOSPHOGLYCAN?) (P) (LIVER? OR PLACENTA?)

=> s l1 and antibod?
        385449 ANTIBOD?
L2           7 L1 AND ANTIBOD?

=> d l2 abs ibib kwic 1-7

L2   ANSWER 1 OF 7  HCAPLUS  COPYRIGHT 2003 ACS on STN
AB   Insulin signaling to generate inositol phosphoglycans
     (IPGs) was demonstrated to occur via the participation of the
     heterotrimeric G-proteins Gq/11.  IPGs were measured as two specific
     inositol markers, myo-inositol and chiro-inositol after strong acid
     hydrolysis.  Insulin and Pasteurella multocida toxin (PMT) generated both
     myo-inositol and chiro-inositol IPGs in a dose-dependent manner.  PMT has
     been shown to activate Gq specifically.  Insulin action was abrogated by
     pre-treatment with anti Gq/11 antibody.  Western blotting
     demonstrated the enrichment of both insulin receptor .beta. subunit and
     Gq/11 in the liver membrane vesicles.  Vesicles also contained
     clathrin, caveolin PLC .beta.1 and PLC.DELTA..  Immunogold staining
     revealed the co-localization of both insulin receptor .beta. subunit and
     Gq/11 in an approx. stochiometric ratio of 1:3.  No vesicles were detected
     with either component alone.  The present and considerable published data
     provide strong evidence for insulin signaling both via a tyrosine kinase
     cascade mechanism and via heterotrimeric G-protein interactions.
ACCESSION NUMBER:        2002:513856  HCAPLUS
DOCUMENT NUMBER:         137:289240
TITLE:                   Gq/11 is involved in insulin-stimulated
                         inositol phosphoglycan putative
                         mediator generation in rat liver membranes:
                         co-localization of Gq/11 with the insulin receptor in
                         membrane vesicles
AUTHOR(S):               Sleight, S.; Wilson, B. A.; Heimark, D. B.; Larner, J.
CORPORATE SOURCE:        Department of Pharmacology, University of Virginia
                         School of Medicine, Charlottesville, VA, 22908, USA
SOURCE:                  Biochemical and Biophysical Research Communications
                         (2002), 295(2), 561-569
                         CODEN: BBRCA9; ISSN: 0006-291X
PUBLISHER:               Elsevier Science
DOCUMENT TYPE:           Journal
LANGUAGE:                English
REFERENCE COUNT:         42    THERE ARE 42 CITED REFERENCES AVAILABLE FOR THIS
                               RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT
TI   Gq/11 is involved in insulin-stimulated inositol
     phosphoglycan putative mediator generation in rat liver
     membranes: co-localization of Gq/11 with the insulin receptor in membrane
     vesicles
AB   Insulin signaling to generate inositol phosphoglycans
     (IPGs) was demonstrated to occur via the participation of the
     heterotrimeric G-proteins Gq/11.  IPGs were measured as two specific
```

inositol markers, myo-inositol and chiro-inositol after strong acid hydrolysis. Insulin and Pasteurella multocida toxin (PMT) generated both myo-inositol and chiro-inositol IPGs in a dose-dependent manner. PMT has been shown to activate Gq specifically. Insulin action was abrogated by pre-treatment with anti Gq/11 **antibody**. Western blotting demonstrated the enrichment of both insulin receptor .beta. subunit and Gq/11 in the **liver** membrane vesicles. Vesicles also contained clathrin, caveolin PLC .beta.1 and PLC.DELTA.. Immunogold staining revealed the co-localization of both insulin receptor .beta. subunit and Gq/11 in an approx. stochiometric ratio of 1:3. No vesicles were detected with either component alone. The present and considerable published data provide strong evidence for insulin signaling both via a tyrosine kinase cascade mechanism and via heterotrimeric G-protein interactions.

ST G protein insulin **inositol phosphoglycan liver** membrane signaling; membrane vesicle **liver** G protein insulin receptor localization

IT G proteins (guanine nucleotide-binding proteins)
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(Gq; Gq/11 in mechanism for insulin-stimulated **inositol phosphoglycan** mediator generation in rat **liver** membranes and localization of G protein with insulin receptor in membrane vesicles)

IT Cell membrane
Signal transduction, biological
(Gq/11 in mechanism for insulin-stimulated **inositol phosphoglycan** mediator generation in rat **liver** membranes and localization of G protein with insulin receptor in membrane vesicles)

IT Clathrin
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(Gq/11 in mechanism for insulin-stimulated **inositol phosphoglycan** mediator generation in rat **liver** membranes and localization of G protein with insulin receptor in membrane vesicles in relation to clathrin)

IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(caveolins; Gq/11 in mechanism for insulin-stimulated **inositol phosphoglycan** mediator generation in rat **liver** membranes and localization of G protein with insulin receptor in membrane vesicles in relation to caveolin)

IT Glycophospholipids
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(phosphatidylinositol-contg; Gq/11 in mechanism for insulin-stimulated **inositol phosphoglycan** mediator generation in rat **liver** membranes and localization of G protein with insulin receptor in membrane vesicles)

IT Insulin receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(.beta. subunit; Gq/11 in mechanism for insulin-stimulated **inositol phosphoglycan** mediator generation in rat **liver** membranes and localization of G protein with insulin receptor in membrane vesicles)

IT 87-89-8, myo-Inositol 9004-10-8, Insulin, biological studies
38876-99-2, chiro-Inositol
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(Gq/11 in mechanism for insulin-stimulated **inositol phosphoglycan** mediator generation in rat **liver** membranes and localization of G protein with insulin receptor in

membrane vesicles)
IT 63551-76-8, Phosphatidylinositol phospholipase C
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(.beta.1 and .delta. isoforms; Gq/11 in mechanism for insulin-stimulated **inositol phosphoglycan** mediator generation in rat **liver** membranes and localization of G protein with insulin receptor in membrane vesicles)

L2 ANSWER 2 OF 7 HCAPLUS COPYRIGHT 2003 ACS on STN
AB A family of A-type inositolphosphoglycans (IPGs) from human **liver** and **placenta** that appear to play a role in the regulation of lipogenesis are identified and characterized. These substances have the biol. activity assocd. with A-type **IPG** fractions, namely regulating lipogenic activity and inhibiting cAMP dependent protein kinase. The characterization of the compds. demonstrates that they contain metal ions, in particular Zn2+, and optionally phosphate. The compds. and their antagonists have uses as pharmaceuticals, e.g. for the treatment of diabetes, and in screening for synthetic analogs.

ACCESSION NUMBER: 1998:180888 HCAPLUS
DOCUMENT NUMBER: 128:242350
TITLE: A type A glycosylphosphatidylinositol second messenger from human tissue involve in regulation of lipogenesis
INVENTOR(S): Rademacher, Thomas William; Caro, Hugo
PATENT ASSIGNEE(S): Hoeft Rademacher Ltd., UK; Rademacher, Thomas William; Caro, Hugo
SOURCE: PCT Int. Appl., 62 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9811116	A1	19980319	WO 1997-GB2444	19970911
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
AU 9741307	A1	19980402	AU 1997-41307	19970911
AU 713103	B2	19991125		
EP 925305	A1	19990630	EP 1997-939087	19970911
EP 925305	B1	20000426		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
AT 192161	E	20000515	AT 1997-939087	19970911
ES 2147996	T3	20001001	ES 1997-939087	19970911
JP 2001504450	T2	20010403	JP 1998-513368	19970911
US 6303580	B1	20011016	US 1999-254797	19990604
US 2001039027	A1	20011108	US 2001-775856	20010201

PRIORITY APPLN. INFO.: GB 1996-18930 A 19960911
WO 1997-GB2444 W 19970911
US 1999-254797 A3 19990604
REFERENCE COUNT: 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS

RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

AB A family of A-type inositolphosphoglycans (IPGs) from human **liver** and **placenta** that appear to play a role in the regulation of lipogenesis are identified and characterized. These substances have the biol. activity assocd. with A-type **IPG** fractions, namely regulating lipogenic activity and inhibiting cAMP dependent protein kinase. The characterization of the compds. demonstrates that they contain metal ions, in particular Zn2+, and optionally phosphate. The compds. and their antagonists have uses as pharmaceuticals, e.g. for the treatment of diabetes, and in screening for synthetic analogs.

IT **Antibodies**
RL: ARG (Analytical reagent use); BSU (Biological study, unclassified); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(monoclonal, to type A glycosylphosphatidylinositol; type A glycosylphosphatidylinositol second messenger from human tissue involve in regulation of lipogenesis)

IT **Antibodies**
RL: ARG (Analytical reagent use); BSU (Biological study, unclassified); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(to type A glycosylphosphatidylinositol; type A glycosylphosphatidylinositol second messenger from human tissue involve in regulation of lipogenesis)

L2 ANSWER 3 OF 7 HCAPLUS COPYRIGHT 2003 ACS on STN

AB Membrane assocd. glycosyl-phosphatidylinositols have been shown to be the precursors of **inositol phosphoglycan** second messengers. Extn. of human **liver** membranes and purifn. by serial thin layer chromatog. revealed three glycolipids which co-migrated with glycosyl-phosphatidylinositol from rat **liver**. These lipidic fractions were partially sensitive to treatment with nitrous acid and to hydrolysis by glycosyl-phosphatidylinositol-specific phospholipase D from bovine serum. In parallel, glycosyl-phosphatidylinositol isolated from rat **liver** was found to be a substrate for the enzyme generating a biol. active **inositol phosphoglycan** species (detd. by measuring inhibition of protein kinase A activity and stimulation of cell proliferation within the chicken embryo cochleovestibular ganglion). This mol. was recognized by an anti-**inositol phosphoglycan antibody**. Hence, we propose that glycosyl-phosphatidylinositol-specific phospholipase D could be implicated in cellular signaling.

ACCESSION NUMBER: 1997:285892 HCAPLUS
DOCUMENT NUMBER: 127:2368
TITLE: Glycosyl-phosphatidylinositol-phospholipase type D: a possible candidate for the generation of second messengers
AUTHOR(S): Jones, David R.; Avila, Matias A.; Sanz, Carmen; Varela-Nieto, Isabel
CORPORATE SOURCE: Instituto de Investigaciones Biomedicas, Consejo Superior de Investigaciones Cientificas, Madrid, 28029, Spain
SOURCE: Biochemical and Biophysical Research Communications (1997), 233(2), 432-437
CODEN: BBRCA9; ISSN: 0006-291X
PUBLISHER: Academic
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Membrane assocd. glycosyl-phosphatidylinositols have been shown to be the precursors of **inositol phosphoglycan** second messengers. Extn. of human **liver** membranes and purifn. by serial thin layer chromatog. revealed three glycolipids which co-migrated with glycosyl-phosphatidylinositol from rat **liver**. These lipidic fractions were partially sensitive to treatment with nitrous acid and to hydrolysis by glycosyl-phosphatidylinositol-specific phospholipase D from bovine serum. In parallel, glycosyl-phosphatidylinositol isolated from rat **liver** was found to be a substrate for the enzyme generating a biol. active **inositol phosphoglycan** species (detd. by measuring inhibition of protein kinase A activity and stimulation of cell proliferation within the chicken embryo cochleovestibular ganglion). This mol. was recognized by an anti-**inositol phosphoglycan antibody**. Hence, we propose that glycosyl-phosphatidylinositol-specific phospholipase D could be implicated in cellular signaling.

L2 ANSWER 4 OF 7 HCAPLUS COPYRIGHT 2003 ACS on STN

AB **Inositol-phosphoglycan** (**IPG**) is a putative mediator of insulin action that has been shown to affect numerous biochem. processes. **IPG**, prepd. from **liver** membranes, promptly inhibited phenylephrine- or vasopressin-induced [Ca2+]i oscillations when perfused over Fura-2-dextran injected rat hepatocytes. An **antibody** to **IPG** suppressed the inhibitory effect of insulin on the [Ca2+]i oscillations. Measurement of the rate of quench of cytoplasmic Fura-2 by extracellular Mn2+ showed that Ca2+ entry occurred continuously in the unstimulated cell and was not affected by phenylephrine or vasopressin. **IPG**, specifically, almost completely abolished the Mn2+ quench rate. Elevated extracellular [Ca2+] reversed the inhibitory effect of **IPG** on [Ca2+]i oscillations. We conclude that **IPG** inhibits the hepatocyte Ca2+ oscillator by reducing the continuous Ca2+ influx that is required to sustain oscillations in [Ca2+]i.

ACCESSION NUMBER: 1997:188451 HCAPLUS
DOCUMENT NUMBER: 126:272579
TITLE: Inositol-phosphoglycan inhibits calcium oscillations in hepatocytes by reducing calcium entry
AUTHOR(S): Sanchez-Bueno, Antonio; Greenwood, Mark R.; Varela-Nieto, Isabel; Marrero, Isabel; Gil, Beatriz; Mato, Jose M.; Cobbold, Peter H.
CORPORATE SOURCE: Dep. Human Anatomy & Cell Biology, Univ. Liverpool, Liverpool, UK
SOURCE: Cell Calcium (1997), 21(2), 125-133
CODEN: CECADV; ISSN: 0143-4160
PUBLISHER: Churchill Livingstone
DOCUMENT TYPE: Journal
LANGUAGE: English

AB **Inositol-phosphoglycan** (**IPG**) is a putative mediator of insulin action that has been shown to affect numerous biochem. processes. **IPG**, prepd. from **liver** membranes, promptly inhibited phenylephrine- or vasopressin-induced [Ca2+]i oscillations when perfused over Fura-2-dextran injected rat hepatocytes. An **antibody** to **IPG** suppressed the inhibitory effect of insulin on the [Ca2+]i oscillations. Measurement of the rate of quench of cytoplasmic Fura-2 by extracellular Mn2+ showed that Ca2+ entry occurred continuously in the unstimulated cell and was not affected by phenylephrine or vasopressin. **IPG**, specifically, almost completely abolished the Mn2+ quench rate. Elevated extracellular [Ca2+]

reversed the inhibitory effect of **IPG** on [Ca2+]i oscillations. We conclude that **IPG** inhibits the hepatocyte Ca2+ oscillator by reducing the continuous Ca2+ influx that is required to sustain oscillations in [Ca2+]i.

IT **Liver**
(hepatocyte; **inositol-phosphoglycan** mediation of insulin action and inhibition of calcium oscillations in hepatocytes)

L2 ANSWER 5 OF 7 HCAPLUS COPYRIGHT 2003 ACS on STN

AB Previous studies on the isoform compn. of human RNases have resulted in confusing and inconsistent results, presumably due to methodol. problems in electrofocusing of alk. proteins. In the present study, immobilized pH gradient (**IPG**) carrier ampholyte (CA) isoelec. focusing (IEF) and conventional CA-IEF have been evaluated for the anal. of the isoforms of human non-secretory RNases purified from kidney, **liver** and spleen. CA-IEF proved unsuitable since the alk. RNase isoforms migrated into the cathode. **IPG**-CA-IEF, however, resolved the RNase isoforms and marker proteins in the basic region of the gel matrix. The three RNases had comparable isoform profiles, each with two protein bands with approx. pI values of 10.3 and 10.4. Western blotting showed that the two protein bands of each RNase were immunoreactive (with polyclonal **antibodies** that recognize RNase), indicating that the protein bands are RNase isoforms. The present results provide reliable pI data on human RNase isoforms and suggest that **IPG**-CA-IEF should be a suitable technique for analyzing the isoforms of other alk. proteins.

ACCESSION NUMBER: 1994:3306 HCAPLUS
DOCUMENT NUMBER: 120:3306
TITLE: Immobilized pH gradient focusing of alkaline proteins: Analysis of the isoform composition of purified human non-secretory ribonucleases from kidney, liver and spleen
AUTHOR(S): Coronel, Elizabeth C.; Little, Brian W.; Alhadeff, Jack A.
CORPORATE SOURCE: Cent. Mol. Biosci. Biotechnol., Lehigh Univ., Bethlehem, PA, 18015, USA
SOURCE: Biochemical Journal (1993), 296(3), 553-6
CODEN: BIJOAK; ISSN: 0306-3275
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Previous studies on the isoform compn. of human RNases have resulted in confusing and inconsistent results, presumably due to methodol. problems in electrofocusing of alk. proteins. In the present study, immobilized pH gradient (**IPG**) carrier ampholyte (CA) isoelec. focusing (IEF) and conventional CA-IEF have been evaluated for the anal. of the isoforms of human non-secretory RNases purified from kidney, **liver** and spleen. CA-IEF proved unsuitable since the alk. RNase isoforms migrated into the cathode. **IPG**-CA-IEF, however, resolved the RNase isoforms and marker proteins in the basic region of the gel matrix. The three RNases had comparable isoform profiles, each with two protein bands with approx. pI values of 10.3 and 10.4. Western blotting showed that the two protein bands of each RNase were immunoreactive (with polyclonal **antibodies** that recognize RNase), indicating that the protein bands are RNase isoforms. The present results provide reliable pI data on human RNase isoforms and suggest that **IPG**-CA-IEF should be a suitable technique for analyzing the isoforms of other alk. proteins.

L2 ANSWER 6 OF 7 HCAPLUS COPYRIGHT 2003 ACS on STN

AB Two **inositol phosphoglycans** were isolated from the

bovine **liver** by chromatog. on AG 1.times.8 ion exchange column and selective elution with HCl at pH 2.0 and 1.3. The pH 2.0 mediator contg. D-chiroinositol stimulated pyruvate dehydrogenase phosphatase, whereas the pH 1.3 mediator contg. myo-inositol inhibited cAMP-dependent protein kinase. Each mediator was further purified by TLC and Bio-Gel P4 column chromatog. and injected i.p. to normal fed rats together with [U-14C]glucose. After 2.5 h, the diaphragms were removed and glycogen isolated. The insulin mediators, like insulin, stimulated the [U-14C]glucose incorporation into glycogen by 150-160% in a dose-dependent manner in the nanomolar range. The mediators injected i.v. in the nanomolar range into low-dose streptozotocin-diabetic rats decreased blood plasma glucose 30-45% in 30-60 min, with a return to basal concns. after 150-180 min. These insulin-like effects were obsd. without changes in blood serum insulin concns. The pH 2.0 mediator was 50-100-times more active than the pH 1.3 mediator in the i.p. diaphragm glycogenesis assay. The mediator effects on the diaphragm were completely blocked by preincubation with an immunopurified **inositol phosphoglycan antibody**. Both mediators were equally active i.v. in lowering plasma glucose at concns. comparable to those of insulin.

ACCESSION NUMBER: 1993:139626 HCAPLUS
DOCUMENT NUMBER: 118:139626
TITLE: Chiroinositol deficiency and insulin resistance. III. Acute glycogenic and hypoglycemic effects of two inositol phosphoglycan insulin mediators in normal and streptozotocin-diabetic rats in vivo
AUTHOR(S): Huang, Laura C.; Fonteles, Manasses C.; Houston, Devin B.; Zhang, Chenggui; Larner, Joseph
CORPORATE SOURCE: Sch. Med., Univ. Virginia, Charlottesville, VA, 22908, USA
SOURCE: Endocrinology (1993), 132(2), 652-7
CODEN: ENDOAO; ISSN: 0013-7227
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Two **inositol phosphoglycans** were isolated from the bovine **liver** by chromatog. on AG 1.times.8 ion exchange column and selective elution with HCl at pH 2.0 and 1.3. The pH 2.0 mediator contg. D-chiroinositol stimulated pyruvate dehydrogenase phosphatase, whereas the pH 1.3 mediator contg. myo-inositol inhibited cAMP-dependent protein kinase. Each mediator was further purified by TLC and Bio-Gel P4 column chromatog. and injected i.p. to normal fed rats together with [U-14C]glucose. After 2.5 h, the diaphragms were removed and glycogen isolated. The insulin mediators, like insulin, stimulated the [U-14C]glucose incorporation into glycogen by 150-160% in a dose-dependent manner in the nanomolar range. The mediators injected i.v. in the nanomolar range into low-dose streptozotocin-diabetic rats decreased blood plasma glucose 30-45% in 30-60 min, with a return to basal concns. after 150-180 min. These insulin-like effects were obsd. without changes in blood serum insulin concns. The pH 2.0 mediator was 50-100-times more active than the pH 1.3 mediator in the i.p. diaphragm glycogenesis assay. The mediator effects on the diaphragm were completely blocked by preincubation with an immunopurified **inositol phosphoglycan antibody**. Both mediators were equally active i.v. in lowering plasma glucose at concns. comparable to those of insulin.

L2 ANSWER 7 OF 7 HCAPLUS COPYRIGHT 2003 ACS on STN
AB New plasma assays for fibrin(ogen) degrdn. products have become available

which are based upon monoclonal **antibodies** and can be performed in plasma. In this study, such specific enzyme immunoassays were evaluated, i.e.: for the total of degrdn. products of fibrin and of fibrinogen (TDP), fibrin degrdn. products (D-dimer and FbDP), and fibrinogen degrdn. products (FgDP) in patients suspected of having deep venous thrombosis of the leg (DVT) and patients with cirrhosis of the **liver**. DVT was assessed by impedance plethysmog. (**IPG**). In each of the (sub)groups of patients, a very good correlation ($0.90 < r < 0.98$) was obsd. between the actually measured TDP values and the calcd. sum of the sep. measured FbDP and FgDP levels. Only 2% (5 patients) of the cases showed a discrepancy of more than a factor two between the found TDP values and the calcd. sum of the measured FbDP and FgDP levels. About 90% of the fibrin degrdn. products were crosslinked. FbDP levels correlated well with the FgDP levels ($0.72 < r < 0.94$) and D-dimer levels ($0.82 < r < 0.91$) in both patients with DVT and cirrhotics. In those patients also, a good correlation ($0.67 < r < 0.83$) was obsd. between FgDP and D-dimer levels, but not in patients suspected of having DVT but with a normal **IPG** test result. Secondary fibrinolysis appeared to be accompanied by fibrinogenolysis.

ACCESSION NUMBER: 1991:202966 HCAPLUS
DOCUMENT NUMBER: 114:202966
TITLE: Correlations between plasma levels of fibrin(ogen) derivatives as quantified by different assays based on monoclonal **antibodies**
AUTHOR(S): Kroneman, H.; Nieuwenhuizen, W.; Knot, E. A. R.; Van Bergen, P. F. M. M.; De Maat, M. P. M.
CORPORATE SOURCE: Dep. Intern. Med. II, Univ. Hosp. Dijkzigt-Rotterdam, Rotterdam, Neth.
SOURCE: Thrombosis Research (1991), 61(4), 441-52
CODEN: THBRAA; ISSN: 0049-3848
DOCUMENT TYPE: Journal
LANGUAGE: English

TI Correlations between plasma levels of fibrin(ogen) derivatives as quantified by different assays based on monoclonal **antibodies**

AB New plasma assays for fibrin(ogen) degrdn. products have become available which are based upon monoclonal **antibodies** and can be performed in plasma. In this study, such specific enzyme immunoassays were evaluated, i.e.: for the total of degrdn. products of fibrin and of fibrinogen (TDP), fibrin degrdn. products (D-dimer and FbDP), and fibrinogen degrdn. products (FgDP) in patients suspected of having deep venous thrombosis of the leg (DVT) and patients with cirrhosis of the **liver**. DVT was assessed by impedance plethysmog. (**IPG**). In each of the (sub)groups of patients, a very good correlation ($0.90 < r < 0.98$) was obsd. between the actually measured TDP values and the calcd. sum of the sep. measured FbDP and FgDP levels. Only 2% (5 patients) of the cases showed a discrepancy of more than a factor two between the found TDP values and the calcd. sum of the measured FbDP and FgDP levels. About 90% of the fibrin degrdn. products were crosslinked. FbDP levels correlated well with the FgDP levels ($0.72 < r < 0.94$) and D-dimer levels ($0.82 < r < 0.91$) in both patients with DVT and cirrhotics. In those patients also, a good correlation ($0.67 < r < 0.83$) was obsd. between FgDP and D-dimer levels, but not in patients suspected of having DVT but with a normal **IPG** test result. Secondary fibrinolysis appeared to be accompanied by fibrinogenolysis.

IT **Antibodies**
RL: ANST (Analytical study)
(monoclonal, in EIA for fibrin and fibrinogen degrdn. products in plasma)

09/775,856

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Delacroix

L2 ANSWER 6 OF 7 HCAPLUS COPYRIGHT 2003 ACS on STN

AB Two **inositol phosphoglycans** were isolated from the bovine **liver** by chromatog. on AG 1.times.8 ion exchange column and selective elution with HCl at pH 2.0 and 1.3. The pH 2.0 mediator contg. D-chiroinositol stimulated pyruvate dehydrogenase phosphatase, whereas the pH 1.3 mediator contg. myo-inositol inhibited cAMP-dependent protein kinase. Each mediator was further purified by TLC and Bio-Gel P4 column chromatog. and injected i.p. to normal fed rats together with [U-14C]glucose. After 2.5 h, the diaphragms were removed and glycogen isolated. The insulin mediators, like insulin, stimulated the [U-14C]glucose incorporation into glycogen by 150-160% in a dose-dependent manner in the nanomolar range. The mediators injected i.v. in the nanomolar range into low-dose streptozotocin-diabetic rats decreased blood plasma glucose 30-45% in 30-60 min, with a return to basal concns. after 150-180 min. These insulin-like effects were obsd. without changes in blood serum insulin concns. The pH 2.0 mediator was 50-100-times more active than the pH 1.3 mediator in the i.p. diaphragm glycogenesis assay. The mediator effects on the diaphragm were completely blocked by preincubation with an immunopurified **inositol phosphoglycan antibody**. Both mediators were equally active i.v. in lowering plasma glucose at concns. comparable to those of insulin.

ACCESSION NUMBER: 1993:139626 HCAPLUS
DOCUMENT NUMBER: 118:139626
TITLE: Chiroinositol deficiency and insulin resistance. III. Acute glycogenic and hypoglycemic effects of two inositol phosphoglycan insulin mediators in normal and streptozotocin-diabetic rats in vivo
AUTHOR(S): Huang, Laura C.; Fonteles, Manasses C.; Houston, Devin B.; Zhang, Chenggui; Larner, Joseph
CORPORATE SOURCE: Sch. Med., Univ. Virginia, Charlottesville, VA, 22908, USA
SOURCE: Endocrinology (1993), 132(2), 652-7
CODEN: ENDOAO; ISSN: 0013-7227
DOCUMENT TYPE: Journal
LANGUAGE: English

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